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Pathophysiological classification of perinatal depressions and cybernetics in obstetrics — a working hypothesis for a model of life

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Since about a decade ago the electronic fetal heart rate (FHR) monitoring developed by several investigators [8, 11, 14, 32] and the fetal capillary-blood analysis (FBA) for the acid-base status studied by Saling [27, 28] and others [2, 20, 36] have both, increased our knowledge of fetal damage in the perinatal period. Certainly there are some particularly dangerous conditions to be overcome by a fetus in vaginal delivery. This paper describes the **author's present classification of perinatal depressions** based on the pathophysiological hypothesis of how a fetus is jeopardized.

1. Presumptions

1.1 A model of fetal circulation

A fetus lives a fish's life in utero. His circulation and respiration are not yet so differentiated as in the adult into separate respective systems. So, very simply, the system of fetal circulation and respiration (oxygenation) is diagrammatically shown in fig. 1. The anatomy is rather complicated but the function itself is quite simple and primitive. Placental **oxygenation is carried out via the umbilical circulation** which is the by-pass of the fetal descending aorta, carrying about half of the cardiac output as Dawes' [9] data suggest.

1.2 Pathogenesis of two kinds of FHR deceleration during labor

Hon [15] has noted three types of FHR decelerations: early, late and variable with uterine contractions (fig. 2), but it is widely known that only the last two are clinically important. As

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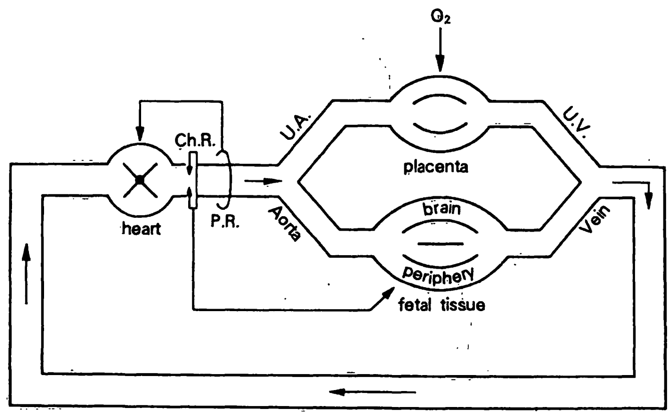


Fig. 1. The simplest **model of fetal circulation and its controlling mechanisms of the heart rate.** U. A. and U. V. are designated for the umbilical arteries and vein. Ch. R. and P. R. mean the chemoreceptors and the presso- or baro-receptors in the fetus. The umbilical circulation carrying out the placental respiration is just a by-pass of the fetal corporeal circuits of aorta.

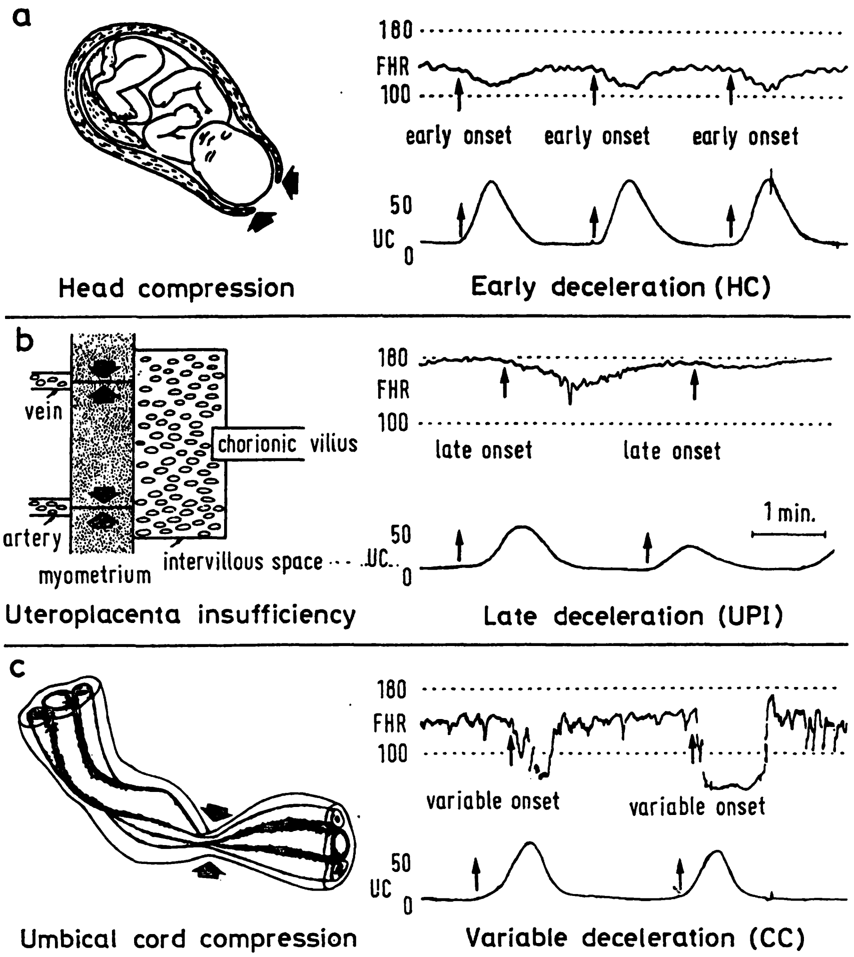


Fig. 2. HON's scheme showing **three types of characteristic FHR deceleration** with uterine contractions, with their physiological mechanisms for each, respectively.

illustrated in fig. 2 the **variable FHR deceleration** is thought to be **induced by such obstructions of the umbilical circulation** as compression or constriction of the cord vessels. According to the model described in fig. 1 such a prompt FHR deceleration can be satisfactorily explained by the **sudden increase of impedance in the umbilical circulation,** which is counteracted by reflex FHR deceleration to keep the blood pressure normotensive. Because about fifty per cent of the total cardiac output is shut out in cases of complete compression of the umbilical arteries, the **FHR must drop** from 140 to 70 or below within a few beats. The physiological experiments and mathematical models by ROSENBLUETH and SIMEONE [26] have shown that such rapid variations in heart rate can only be achieved by **vagal reflex.** The vagus is activated by various afferent stimuli, the most important being the baroreceptive stimuli mediated by **pressure sensors,** such as those in the carotid sinus, as is well documented [5, 13, 18, 19, 34].

In any case, the variability of FHR patterns in "cord" type-decelerations, as far as frequency is concerned, can only be explained by rapid vagal control of heart rate.

On the other hand, if we consider increased impedance in the other half of fetal circulation, that is, in the fetal corporeal blood flow, it is easy to understand how the late FHR deceleration occurs. HON called this a **utero-placental insufficiency (UPI) pattern of FHR deceleration** with uterine contraction, which means that some impediments in the uteroplacental circulation may be the cause of this type of fetal bradycardia. By continuous measurement of P_{O_2} CALDEYRO-BARCIA, POSEIRO, MENDEZ-BAUER and GULIN [7] and WALKER, PHILLIPS, POWE and WOOD [33] showed that some **P_{O_2} fluctuation** quite similar to the UPI pattern occurred in the fetus during uterine contractions. Therefore, the fetal hypoxia inevitably induced during reasonably intense uterine contraction **is signaled through fetal chemoreceptors** (as reported by PURVERS and

BISCOE [24]) and brings about **peripheral vasoconstriction,** resulting in the gradual **increase of vascular impedance.** This is counteracted by the gradual and delayed slowing of **late FHR deceleration (dip II or UPI).** The model in fig. 1 describes this process, enabling us to see how the dynamic dimensions of two types of FHR deceleration can be differentiated from each other. The induction of UPI is caused not only by a single loop of **baroreceptic control** as in "cord" but also by a **chemical loop, via fetal chemoreceptors,** which has a longer reaction time. The primary, physical **emergency reflex** in the circulation is promptly activated by **baroreceptic controls** but the **secondary adaptation** in hypoxic emergency is a redistribution of blood flow, keeping the central organs such as brain and heart supplied, with some sacrifice to non-vital organs such as skin, intestine and extremities (tab. 1).

Circulatory insufficiency of the placenta
due to uterine contraction,
maternal hypotension,
exercise of the mother, etc.
↓
Fetal hypoxia
↓ ← Chemoreceptor reflex
Fetal peripheral vasoconstriction
in the skin, extremities, and intestine.
↓ ← Baroreceptor reflex
Gradual fetal bradycardia
to keep the cerebral and coronary blood pressure normotensive, and probably to save oxygen consumption as a whole.

Tab. 1. A **hypothetical mechanism of late FHR deceleration.** Refer to the model of fetal circulation described in fig. 1.

2. Classification of perinatal depressions

2.1 Acute fetal distress

Upon the basis of a model of fetal circulation and the pathogenesis of fetal bradycardia, it should be easy to understand that there are **three different types of fetal distress** as schematically drawn in fig. 3. The **umbilical blockade** of fetal oxygenation causes sudden **decrease of P_{O_2}** and rapid **retention of carbon dioxide** in the fetal blood,

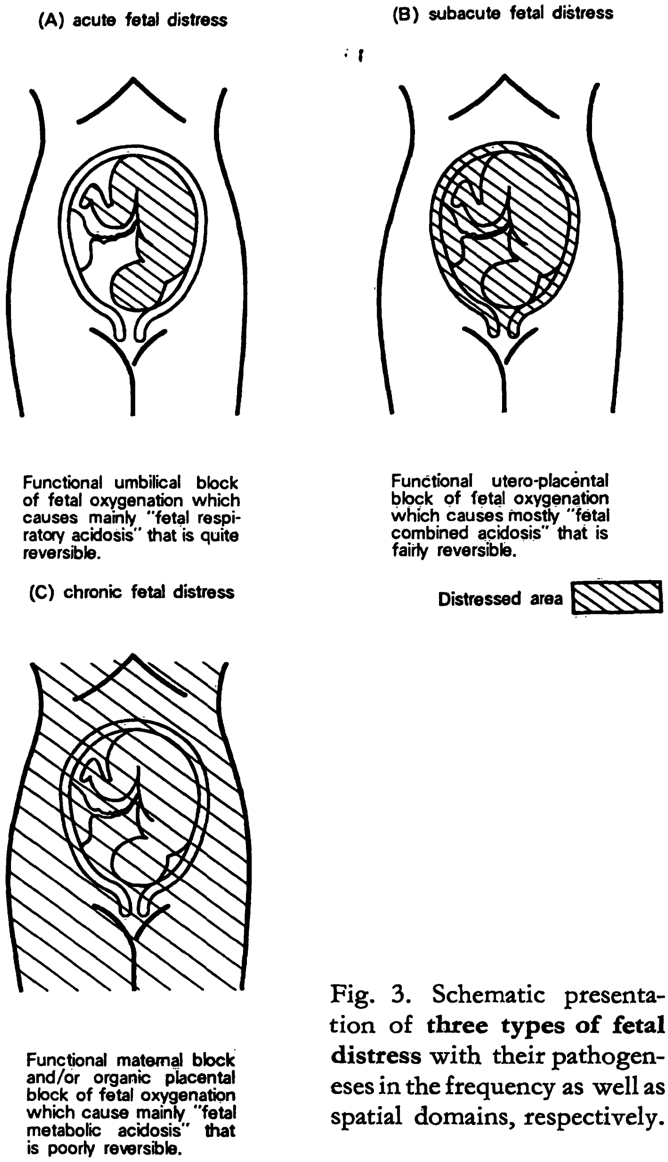


Fig. 3. Schematic presentation of **three types of fetal distress** with their pathogeneses in the frequency as well as spatial domains, respectively.

which in turn brings about **acute, primary respiratory acidosis of the fetus.**

Such a typical case is presented in fig. 4, which shows prolonged marked **fetal bradycardia** (almost as slow as 60 bpm) of about five minutes duration just before delivery. The FBA P_{O_2} was lowered from 20 to 2 mmHg and P_{CO_2} was raised from 38 to 63 mmHg. The **fetal acidosis** of pH 7.15 is respiratory rather than metabolic. Although the acidosis of the umbilical blood was not so pronounced as in the last FBA specimen, the great arterio-venous differences in pH and in P_{O_2} and P_{CO_2} are good etiological evidence for the presence of blockade in the umbilical circulation. The infant was born with a slight depression of 7—9 points (APGAR score) at 1—5 minutes after birth, as evidenced by the transient but marked tachycardia and gradual slow-down in the neonatal heart rate patterns. Acute fetal distress in this case seemed to be caused by a rotation forceps operation impeding the circulation of the **cord which was wound around the neck.**

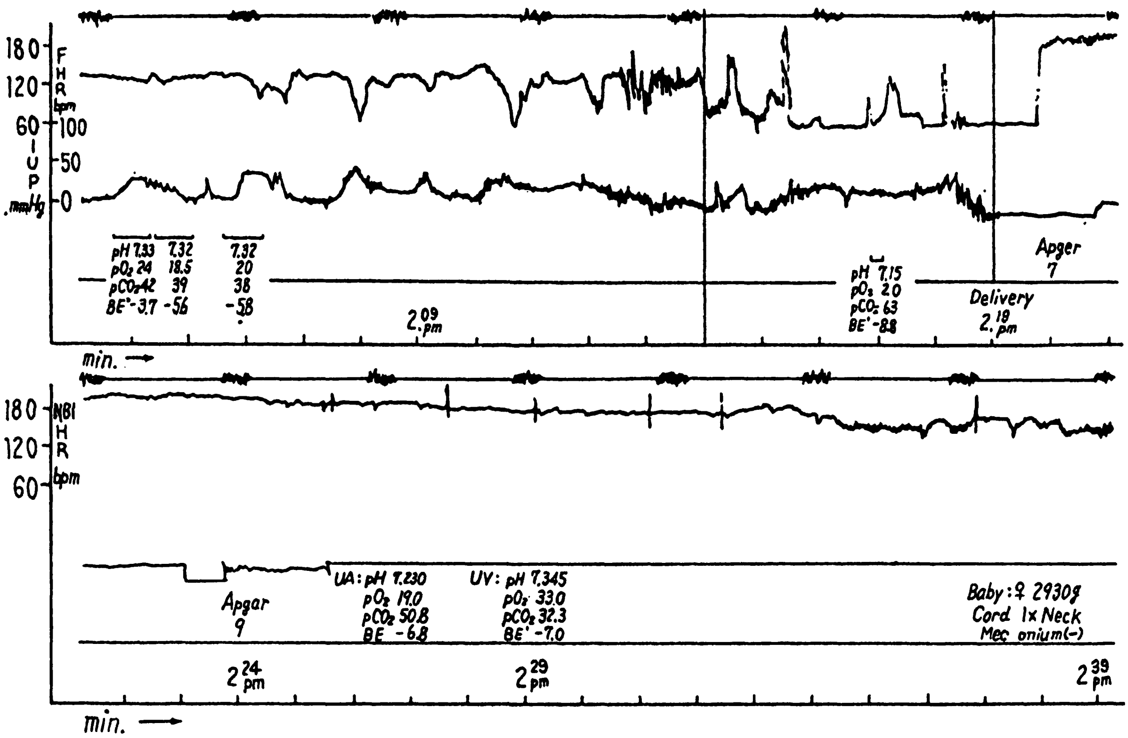


Fig. 4. A typical course of **acute fetal distress** with primary, acute respiratory acidosis in the fetal capillary blood within 5 minutes just before delivery.

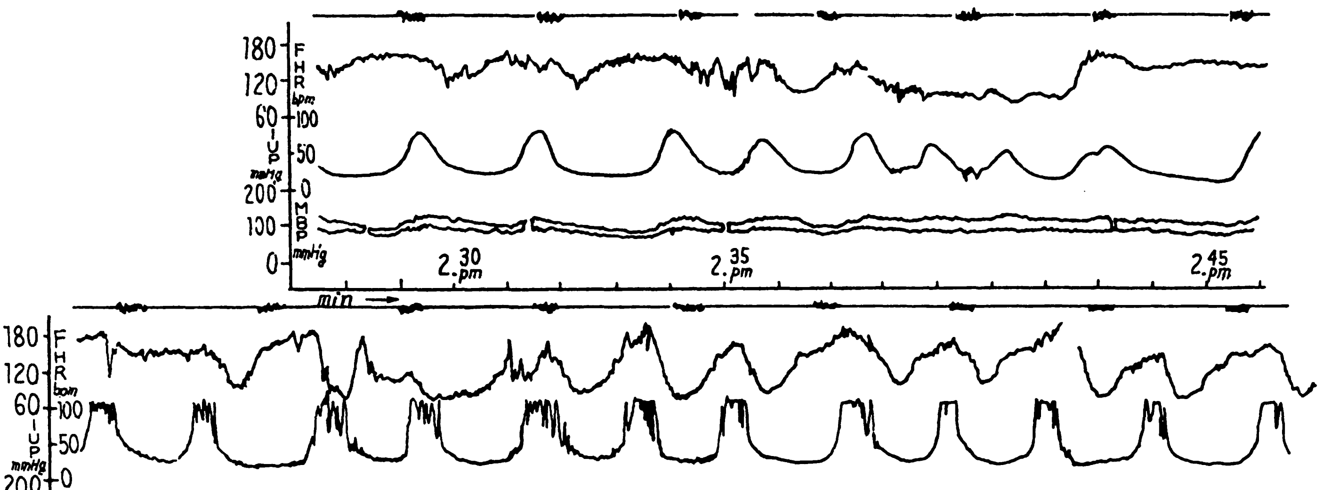


Fig. 5. Quite a typical case of **subacute fetal distress** with combined (respiratory as well as metabolic) acidosis reflecting the whole uterus ischemia induced by maternal hypotension and by tetanic uterine contractions. Note that such marked appearance of late FHR deceleration is to be called "bradycardia paradoxa" on the middle tracing.

2.2 Subacute fetal distress

The fetal oxygenation is also impeded by extra-amniotic, **uteroplacental ischemia,** which is usually caused by tetanic uterine contractions [7, 12] or by a decrease of systemic blood pressure in the mother [4, 16]. The hatched area in fig. 3 (B) covers not only the fetus but the placenta and the uterus.

A typical case, such as demonstrated in fig. 5, showed **both** causes of **insufficient fetal oxygenation.** At first (top, left, third tracing), the maternal blood pressure dropped as low as 80 mmHg systolic; in addition, hypertonic, tachysystolic, tetanic contractions appeared to result in prolonged moderate fetal bradycardia, which was normalized fairly quickly after correction of the hypotension and after the interval between the contractions had become longer. After a while, in the late second stage of labor, the recurrence of dystocic uterine contractions with strong bearing-down elevated the intrauterine pressure markedly and caused enhanced uterine ischemia, resulting in typical late FHR decelerations (UPI) called **"bradycardia paradoxa"** [30], i. e., the FHR accelerates with contraction and decelerates with relaxation. The 1 and 5 minute APGAR scores were 2 and 5, and pronounced fetal acidosis was present in the umbilical blood. The P_{O_2} and base excess are both moderately lowered, so that a **combined, respiratory as well as metabolic acidosis** was present in utero. Frequent and severe **attacks of hypoxia** in the **fetus** and in the **placenta** and **uterine muscles** are probably the cause of this.

2.3 Chronic fetal distress

Another type of fetal distress, when the **whole body** is depressed, can be induced by some **complications in the mother.** Severe **cardio-pulmonary complications,** such as cyanotic heart disorders, or **heavy smoking** are the cause of maldevelopment of the fetus [6], who might not have sufficient oxygen reserves or tolerance to withstand the stress of vaginal delivery. **Toxemia of pregnancy** is also a fairly dangerous complication for the mother as well as for the fetus. The high incidence of small-for-date babies in toxemic patients suggests that some nutritional as well as metabolic disorders are present in the fetus and the placenta. Therefore, even without any predominant changes of the fetal heart rate or other cardio-vascular parameters, such a long-standing fetal or feto-maternal hypoxia may result in **chronic, primary metabolic acidosis of the fetus.**

Fig. 6 describes a typical case of chronic fetal distress, followed by severe asphyxia neonatorum with APGAR scores 1—4. Until the delivery no remarkable FHR changes were noticed, but when we observed carefully, a very slight degree of **late FHR deceleration** was seen consistently with every contraction. The **loss of irregularity of the beat-to-beat FHR variations** was also found. Nevertheless the fetal capillary blood pH had been astonishingly depressed (as low as 7.10 or lower) and combined with a marked reduction of base excess from the beginning. This is one of those rare cases in which there was an enhanced metabolic acidosis in utero without any sign in the FHR except for the slight but quite consistent and insidious appearance of late FHR decelerations from the very early stages of labor.

3. Cybernetic view of the predominant pathogenesis of perinatal depressions (tab. 2)

As shown in fig. 3, these three types of fetal distress correspond to their own spatial as well as frequency domains in the genesis of hypoxia in utero.

Acute fetal distress means acute hypoxia and respiratory acidosis in the fetus due to the impairment of umbilical circulation. These conditions can be induced and corrected within several **minutes.**

Subacute fetal distress due to recurrent blockade of utero-placental circulation cannot kill the fetus so fast but it impairs the neonate if such a process lasts from a half to several **hours** during labor.

Chronic and long-standing hypoxia with primary metabolic acidosis in the fetus may involve a longer process of functional maternal blockade

1. Essential Depression (seconds):
   Neurogenic dysfunction in the onset of respiratoin
2. Acute Depression (minutes):
   Dysfunction of umbilical circulation
3. Subacute Depression (hours):
   Dysfunction of placental oxygenation
4. Chronic Depression (days):
   Metabolic dysfunction of the fetus
5. Organic Depression (months):
   Dysfunction due to malformation and/or maldevelopment

Tab. 2. **Classification of perinatal depressions** according to the predominant pathogenesis with a particular dimension in the spatial as well as in the frequency domains.

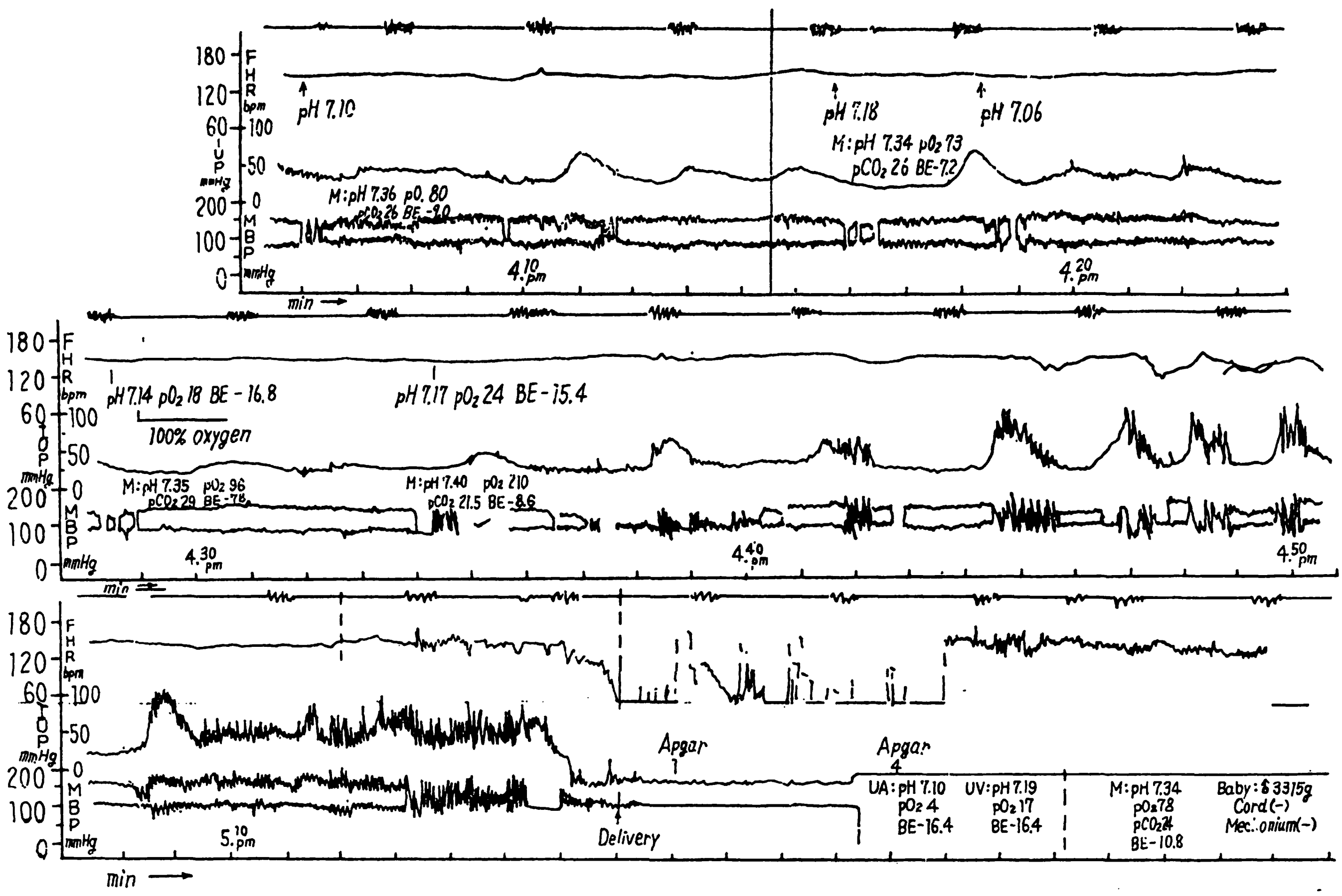


Fig. 6. One of the most typical cases of **chronic fetal distress** complicated by toxemia of pregnancy. Note no particular FHR variations until delivery except quite consistent appearances of slightest degree of late FHR deceleration at every contraction. The fetus had primarily enhanced metabolic acidosis and became depressed with Apgar score 1—4 at one and five minutes after birth. Another finding of the FHR curves is complete loss of beat-to-beat irregularity.

and/or organic placental blockage of fetal oxygenation and nourishment. An exposure of at least several days is needed to so weaken the fetus that the stress of labor will cause irreparable damage. But even with these three conditions no complete explanations can be given for all of the pathogeneses of perinatal depressions. Without any signs of fetal acidosis or FHR variations, some infants do not start pulmonary respiration within a few **seconds** as vigorously as expected. **Respiratory depressants** such as narcotics should be taken into account in these cases of neurogenic dysfunction in the onset of respiration, socalled **"essential depression".**

Others begin to show the symptom of respiratory grunting with some macroscopic malformations or maldevelopment. These infants with anencephaly, esophagotracheal fistula, diaphragm hernia-tion or severe cardiac malformations, developed in the course of several **months** of pregnancy, cannot cry so actively as normal babies; this state is called **"organic depression".**

4. A model of life in view of perinatal depressions

In any system the whole body is only controllable by an "across" variable, potential or pressure. The authority of a government can rule a nation. The supply and transport of electricity, city water and fuel gas are all controlled by their respective pressures and peripherally they are measured in quantity by the respective "through" variables, which are flows. The same is true for blood circulation so that **blood pressure** is one of the most important parameters in the body.

In fig. 7 we show a **model of the circulation** which we built for the simulation study of heart rate control dynamics seen in respiratory sinus arrhythmia [23, 29]. The inspiratory transient of heart rate acceleration may well be explained by the single loop of baroreceptic vagal control of circulation, but the shifting of the level of the controlled variable, blood pressure, cannot be achieved without some contribution of parametric controls by the sympathicus, whose enhancement of cardiac contractility and of peripheral vasoconstriction is indispensable but can never be so prompt as vagal control. The sympathetic loop is slower and longer in the frequency domain [35] and is larger in the spatial domain of its influence. It acts on the vagal control loop paramertically, forming a secondary feedback loop network. Thus a **multiple loop control system** is established, at least for **circulation dynamics.**

A living human body is much more complicated, with many subsystems other than **circulation,** such as the **nervous, respiratory,** digestive or **metabolic,** and **organic systems.** At birth an infant should adapt all of these systems to extra-uterine life, but impairments in any one of the subsystems do not allow this. **Essential depression** of the neonate is brought about within a few seconds after birth by the dysfunction or maladaptation of the nervous system in the onset of pulmonary respiration. **Organic depression,**

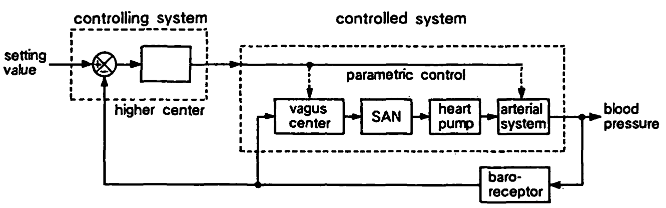


Fig. 7. An example of **multiple loops control in the circulatory system** with parametric control mechanisms of higher sympathetic centers upon the rapidly responsive, vagal control of the heart.

particularly some life-threatening anomalies, cannot be caused in several hours but is the result of months-long pregnancy.

Considering the time dimension (in view of how fast the infant's life is established or destroyed at birth), we can now build a **model of life** (fig. 8) as a multiple loops' feedback system with **five subsystems — nervous, circulatory, respiratory, metabolic** and **organic,** each of which corresponds to our classification of perinatal depressions.

The **nervous system** functions through the prompt transmission of electrical signals. Without

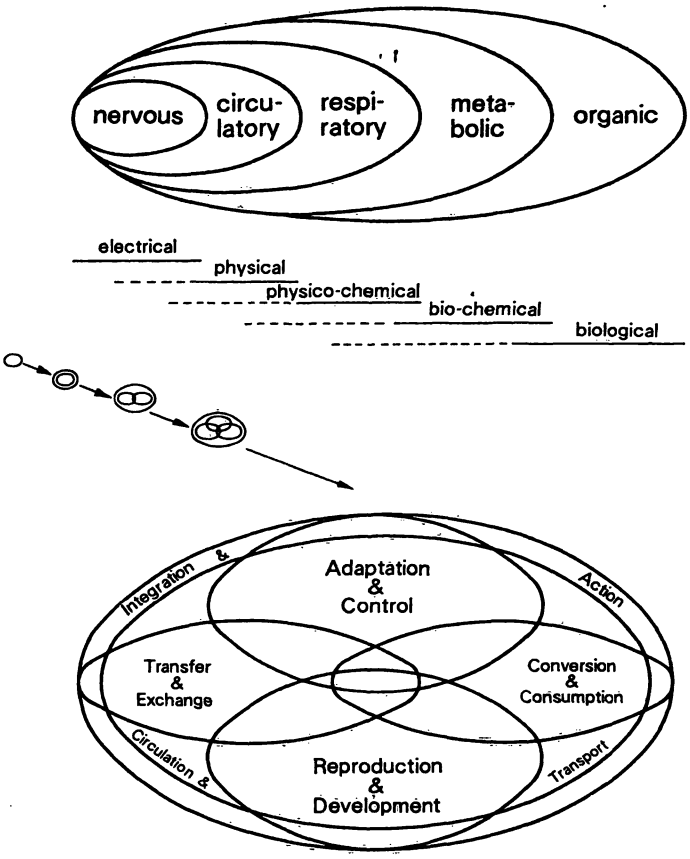


Fig. 8. A mechanical, **steady-state model of life,** functioning as a multiple loops' feedback control system with each particular signal and dimension in the frequency as well as spatial domain for the respective subsystems. The lower half suggests the **biologically dynamic model of life** including the subsystem of reproduction and development.

knowing anything about neural potentials and/or brain waves, it is impossible to discuss how the brain and the nervous system function.

On the other hand, the most important parameters of **circulation** are physical variables, such as **blood pressure** and **flow,** although they are closely connected to the faster nervous system by an electrical signal, ECG, as well as to the slower respiratory system by physicochemical parameters such as arterial blood P_{CO_2}.

Respiration is controlled mostly by Pa_{CO_2} physicochemically [10] and is connected to the larger digestive and metabolic system by the pH of the arterial blood.

The **organic system** of a living body should be studied for the shape (morphology), the reproductive ability (genetics) and for the interaction with other molecules and organisms (pharmacology, immunology and bacteriology).

5. Comment

There has been some confusion about which is better as a controlling parameter of childbirth, fetal pH or FHR patterns [1, 17, 28]. Now, however, according to the model and the classification of perinatal disintegrating processes of life, it is clear that the **metabolic disorders in the fetus cannot be detected by only one of the circulatory parameters, namely the fetal heart rate,** and that the **chemical parameters of the blood such as pH can never give as quick detection of the acutely depressed infant** as that provided by the FHR response [3, 21, 25]. As for perinatal depression, we have already well documented the fact that both early detection by the FHR and sure diagnosis by the pH **together promise better labor monitoring** [31]. The **fetal heart rate** is not only a **parameter of circulation** but also of **placental respiration** in connection with the umbilical by-pass. But we must refrain from an over-evaluation of the FHR because the direct measurement of **pH** tells us how placental oxygenation is being carried out. On the other hand, **the best time for FBA cannot be determined without continuous monitoring of the FHR from the earliest possible stage of labor.** FHR monitoring in the late second stage of labor can provide beat-to-beat information so dynamically that the physician can avoid acute depression just before birth. But it is not entirely reliable since a normal FHR may be recorded even in cases of a severe metabolic acidosis which has endangered the fetus since the very early stages of labor. FHR monitoring and measurements of fetal blood pH in the diagnosis of fetal acidosis can be compared to the smear test and the biopsy in the diagnosis of carcinoma of the uterine cervix. **A diagnosis of all perinatal depressions cannot be made by considering only one of the fetal parameters during labor.** It is better to know the dynamic diagnostic spectrum of each of the parameters well, and to use these parameters accordingly.

Summary

Fetal monitoring during labor and dynamic system analysis of the theoretical physiological basis have both made it possible to build a **model of fetal circulation which can explain the mechanisms of fetal heart rate decelerations** during uterine contractions and also make it possible to **classify perinatal depressions in five categories. Fetal circulation and respiration** are not yet as differentiated as in the adult. So the system of fetal circulation and respiration is modelled as a simple **parallel network,** corporeal and placental, as described in fig. 1. About half of the fetal cardiac output goes to the placenta via the umbilical circulation, which is a by-pass of the fetal corporeal circulation. The **very fast, baroreceptic control system of the vagus** and the **rather slow, chemoreceptic control mechanism which is mainly due to the sympathicus** are both described. According to these assumptions, the **cord type of variable FHR-deceleration** is induced by **activation of baroreceptors** in the fetus due to a sudden increase of hemodynamic impedance in the umbilical circulation (fig. 2). The **UPI or dip II type of late FHR deceleration** is caused by **chemoreceptor stimulation** which in turn causes a sympathetic, gradual increase of vascular impedance in the fetal corporeal circulation; this sacrifice of non-urgent tissues, such as fetal skin, muscles and intestines, results in a slowing down of the fetal heart rate and in a redistribution of the blood, mostly to the heart and brain, thus conserving oxygen and energy consumption as much as possible.

Therefore, whereas the former, **reflex bradycardia,** is a primary self-defense mechanism activated by a prompt single loop ~~feedback in a~~ physical circulatory emergency, the latter, **hypoxic bradycardia,** is a **secondary self-defense mechanism** in hypoxic emergency with another slower chemical loop feedback activated in addition to the former.

So, in cases of such cord complications as prolapse, true knot, multiple loops, over-twisting, and too short or too long and/or too thin a cord, the tight, protracted blockade of umbilical circulation may induce **"acute fetal distress"** with the symptoms of typical **variable FHR decelerations,** such as **marked prolonged fetal bradycardia** of sudden onset, and with those of purely **respiratory acidosis** of the fetus, both of which can be quite reversible but yet may kill the fetus in ten minutes or so, depending on the state of the fetus (figs. 3 and 4).

On the other hand, tetanic uterine contractions, maternal hypotension and physical or psychological excitement may **reduce the utero-placental blood flow,** so that ischemia or hypoxia occurs, not only in the uterus itself, but in the amniotic fluid, the placenta and the fetus, and may induce **combined fetal acidosis (respiratory as well as metabolic)** together with the quite typical and pronounced appearance of **late FHR decelerations.** These are fairly reversible and are accompanied by marked rebound tachycardia between contractions, especially after oxygen administration (fig. 5). The author calls this **"subacute fetal distress",** because it takes from a half to several hours for the development or the correction of this condition.

In addition, in cases of **severe maternal complications, such as toxemia and cardio-pulmonary disorders,** with or without organic changes of the placenta, **"chronic fetal distress"** frequently develops with signs of mild or moderate late FHR decelerations on a slightly elevated baseline of diminished irregularity. These appear rather insidiously but consistently with every contraction from the early stages of labor and are accompanied by **primary metabolic acidosis** of the fetus which is poorly reversible (fig. 6).
Furthermore, regardless of the presence of intrauterine acidosis or bradycardia during labor, some neonates do not cry as vigorously as expected because of **severe malformations or serious birth injuries;** these are the cause of **"organic depression"** in the perinatal period. Others, who develop **apnea and perinatal depression with no obvious etiology,** may be considered as having **"essential depression"** of the respiratory centers of the fetus. This is mostly due to narcotic depressants or to vagal shock, which occurs within a few seconds after birth.
In other words, according to how fast a fetus can be jeopardized, we now have a **classification of perinatal depressions in five categories: essential, acute, subacute, chronic** and **organic.** These are induced in seconds, minutes, hours, days, and months, respectively. Moreover, every one of these corresponds to the dysfunction of one of the multiple loop feedback control systems of life (figs. 7 and 8): the **nervous, circulatory, respiratory, metabolic** and **organic subsystems,** each of which is to be tested by their own specific electrical, mechanical, physico-chemical, biochemical and biological parameters. Therefore, it is easy to understand that **neither the fetal capillary-blood pH nor the fetal heart rate alone can cover the entire diagnostic spectrum of perinatal depressions,** because each feedback loop has a different dynamic dimension in its deterioration and recovery with respect to one of the five subsystems of life.

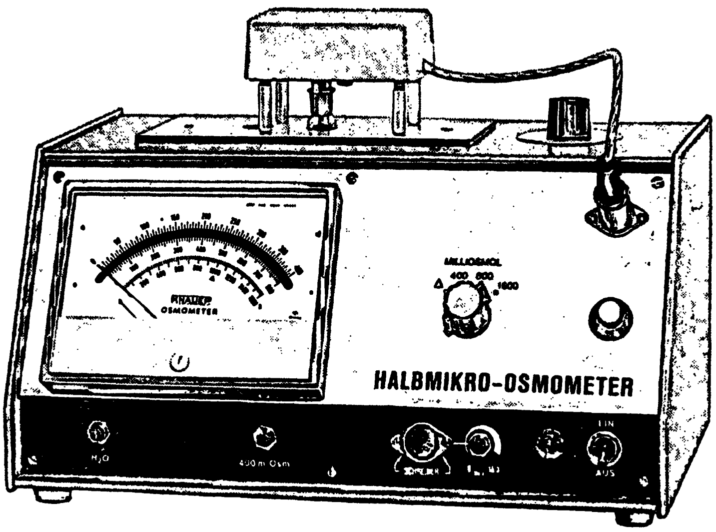
Keywords: Fetus, circulation, acid-base-balance, steady-state, deceleration, fetal distress, perinatal period, depression, cybernetics.

Zusammenfassung

Pathophysiologische Klassifizierung perinataler Depressionen und kybernetische Aspekte in der Geburtshilfe — eine Arbeitshypothese für Regelungsvorgänge im Bereich des Lebendigen.
Anhand der Erfahrung mit hunderten von Feten, die während der Geburt überwacht wurden und bei welchen die Methode der dynamischen Systemanalyse angewandt wurde, konnte ein Modell des fetalen Kreislaufes entwickelt werden, welches es ermöglicht, fetale Pulsdezelerationen während der Wehen zu erklären und mit dessen Hilfe **perinatale Störungen in 5 pathophysiologische Kategorien eingeteilt** werden können.
Da Atmung und Kreislauf des Feten noch nicht so wie beim Erwachsenen differenziert sind, kann der **Kreislauf** als parallelgeschaltetes System — Körper- und Plazentarkreislauf — betrachtet werden (Abb. 1). Etwa die **Hälfte des fetalen Minutenvolumens wird zur Plazenta** durch die Nabelschnurgefäße **geleitet;** dies stellt eine Umgehung des fetalen Körperkreislaufes dar.
Die dynamische Systemanalyse erlaubt die Betrachtung der **Geschwindigkeit,** mit welcher das Pulsfrequenzkontrollsystem bezüglich **Frequenz** und **Phase** reagiert. In diesem Zusammenhang werden die Begriffe des **schnellen Barorezeptorkontrollsystems (Vagus)** und des **langsameren Chemorezeptorsystems (Sympathicus)** eingeführt. Auf Grund dieser Annahmen würde der Nabelschnurtypus der **variablen FHF-Dezeleration durch die Aktivierung von fetalen Barorezeptoren** durch den plötzlichen Anstieg des hämodynamischen Widerstandes im Nabelschnurkreislauf hervorgerufen (Abb. 2).
Der **„dip II"** oder **UPI-Typus** (UPI = utero-plazentare Insuffizienz) der späten FHF-Dezeleration kann dann als **durch Chemorezeptorstimulation bedingt** erklärt werden, welche einen sympathischen, langsamen Anstieg von Gefäßwiderständen im fetalen Körperkreislauf hervorruft unter Vernachlässigung weniger wichtiger Gewebe, wie Haut, Muskel oder Darm. Es ergibt sich dann eine Herzschlagverlangsamung und **Neuverteilung des Blutes in die zentralen Organe (Herz, Gehirn)** und eine **Sparschaltung des gesamten Sauerstoff- und Energieverbrauches.**
Eine Reflexbradykardie ist also ein **primärer Abwehrreflex mit direkter Rückkopplung** in einer Kreislaufnotsituation, während die **Hypoxiebradykardie ein sekundärer Abwehrmechanismus** ist, in welchem die langsamere, **chemische Rückkopplung** zur ersteren hinzugefügt ist.
Dementsprechend kommt es in Fällen von **Nabelschnurkomplikationen** (Vorfall, Knoten, Schlingen, Verdrehung, zu kurze, zu lange oder zu dünne Nabelschnur) zur anhaltenden **Blockierung der Nabelschnurdurchblutung** und **„akuten fetalen Störung"** mit typischen variablen **FHF-Dezelerationen** und anhaltender **Bradykardie** des Feten sowie einer **respiratorischen Azidose.** Beide sind reversibel, können aber je nach dem Zustand des Feten innerhalb von 10 Minuten zum Tode führen (Abb. 3, 4).
Andererseits können tetanische Wehen, mütterliche Hypotension und physische oder psychische Aufregung die **Durchblutung des Uterus reduzieren,** so daß Ischämie und Hypoxie von Fruchtwasser, Planzenta und Fet eine **kombinierte (respiratorische und metabolische) Azidose** hervorruft, die mit typischen späten FHF-Dezelerationen einhergeht. Diese sind einigermaßen **reversibel,** da im Wehenintervall eine Tachykardie besteht, besonders nach Sauerstoffgabe (Abb. 5). Dieser Typ wurde **„subakute fetale Störung"** benannt, da das Leben des Feten erst nach einer halben Stunde oder einigen Stunden bedroht ist.
Außerdem entwickelt sich häufig eine **chronische fetale Störung** mit oder ohne organische Plazentaveränderungen bei **ernsten mütterlichen Komplikationen wie Toxi-**

KNAUER

ELECTRONIC SEMI-MICRO OSMOMETER



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kose oder **Herz- und Lungenkrankheiten,** wobei leichte oder mäßige **späte FHF-Dezelerationen** auf einer etwas **höheren Basisfrequenz** mit **verminderten Schwankungen** bestehen. Diese erscheinen langsam, jedoch **ständig mit jeder Wehe vom Beginn der Wehentätigkeit an.** Es besteht dabei hauptsächlich eine **metabolische Azidose des Feten,** welche schwer zu beheben ist (Abb. 6).

Weiterhin gibt es Neugeborene, welche, ohne daß ein Zusammenhang mit einer intrauterinen Azidose oder Bradykardie besteht, weniger lebhaft als erwartet schreien und bei denen dann eine schwere **Mißbildung** festgestellt wird, wie z. B. Anencephalie, Zwerchfellhernie, Oesophagotrachealfistel, Herzfehler oder Hirnblutung. Bei ihnen wird eine **„organische Depression"** während der Perinatalperiode angenommen. Wieder andere Neugeborene haben eine Apnoe oder eine perinatale Depression **ohne erkennbare Ursache;** diese Depressionen werden als **„essentielle Depression"** des fetalen Atemzentrums durch Narkose oder Vagusschock angesehen; dies ereignet sich innerhalb weniger Sekunden nach der Geburt. Demgemäß ergibt sich aus den dynamischen Merkmalen des Zeitraumes, in welchem der Fet bedroht ist, eine **Klassifizierung der perinatalen Störungen in 5 Kategorien: essentiell, akut, subakut, chronisch** und **organisch,** welche dementsprechend innerhalb von Sekunden, Minuten, Stunden, Tagen oder Monaten auftreten können und dementsprechned behandelt werden müssen. Jede entspricht einer Störung in einem der biologischen Mehrfach-Rückkopplungssysteme (Abb. 7, 8), nämlich des Nerven-, Kreislauf-, Atmungs-, Stoffwechsel- und organischen Subsystems. Diese müssen mit den ihnen entsprechenden, spezifischen Signalen ausgewertet werden, nämlich elektrischen, mechanischen, physikalisch-chemischen, biochemischen und biologischen Parametern. **Es ist daher verständlich, daß weder das pH im fetalen Kapillarblut noch die fetale Herzfrequenz allein das gesamte diagnostische Spektrum perinataler Depressionen umfassen kann,** da jeder Rückkopplungskreis eine andere dynamische Dimension bezüglich Verschlimmerung und Erholung in einem der 5 Lebenssubsysteme hat.

Schlüsselworte: Fetus, Herzschlagregistrierung, Kybernetik, Säure-Basen-Haushalt, Perinatalperiode, Depression, Dezeleration.

Resumé

Classification physiopathologique des dépressions périnatales et cybernétique en Obstétrique — Une hypothèse de travail pour un modèle de vie.

Les expériences de centaines de cas de surveillance foetale pendant le travail, pour la pratique, et la méthode dynamique d'analyse des bases physiologiques, pour la théorie, ont toutes deux conjointement permis d'élaborer un **modèle de la circulation foetale** qui peut expliquer les **mécanismes des décélérations du rythme cardiaque foetal liées aux contractions utérines** ainsi que de **classifier les dépressions périnatales en 5 catégories** selon le mécanisme physiopathologique par lequel un foetus peut être déprimé à la naissance.

La **circulation** et la **respiration** foetales ne sont pas aussi différenciées que chez l'adulte pour avoir des systèmes respectifs distincts.

Aussi, en ce qui concerne la fonction elle-même, le système de la circulation et de la respiration foetale est organisé comme un simple réseau parallèle, foetal et placentaire (fig. 1). Car, à peu près la moitié du débit cardiaque foetal va au placenta par la circulation ombilicale qui court-circuite la circulation corporelle foetale.

En vue d'une analyse d'un système dynamique concernant la rapidité avec laquelle le rythme cardiaque contrôle les réponses de l'organisme en fréquence et en phase, 2 **systèmes doivent être introduits:** un, **très rapide, le système de contrôle baro-récepteur du vague efférent;** un, **plutôt lent, le mécanisme de contrôle par les chémo-recepteurs, principalement dû au sympathique.**

Selon ces présomptions, **le type de ralentissement du rythme cardiaque foetal** par compression funiculaire est **induit par l'activation des baro-récepteurs** chez le foetus, **lié à un brusque accroissement de l'entrave hémodynamique** dans la circulation ombilicale (fig. 2).

Le type «insuffisance utéro-placentaire» ou dip II (décélération tardive), est expliqué comme déterminé par la **stimulation des chémo-récepteurs** qui entraîne un **accroissement progressif, sympathique,** des impédances vasculaires dans la circulation corporelle foetale, **sacrifiant les tissus non urgents** comme la peau, les muscles et les intestins du foetus pour obtenir le **ralentissement du rythme cardiaque foetal** et la **redistribution du sang principalement pour les organes centraux,** coeur et cerveau, préservant la consommation d'oxygène et d'énergie autant que possible dans sa totalité.

Ainsi donc, tandis que **le premier réflexe de bradycardie est un réflexe primaire d'autodéfense,** par un circuit rapide, unique de **feedback physique dans l'urgence circulatoire, le tardif, la bradycardie hypoxique est un mécanisme secondaire d'autodéfense dans l'urgence hypoxique** par un autre circuit de **feedback chimique activé plus lentement** en adjonction au premier.

Ainsi dans les cas de différentes complications du cordon comme la procidence, un noeud vrai, des torsions circulaires et brièreté, le blocage serré prolongé de la circulation ombilicale peut induire **«la détresse foetale aiguë»** (fig. 3) avec comme marques, les symptomes de **décélérations variables typiques,** une **bradycardie foetale prolongée d'apparition soudaine** ainsi qu'une **acidose purement respiratoire du foetus,** chacune des deux pouvant être entièrement **réversibles** mais cependant **pouvant tuer le foetus en 10 minutes ou plus,** dépendant de la part de souffrance chronique du foetus.

D'un autre coté, des **contractions utérines tétaniques,** une **hypotension maternelle** et une excitation physique ou psychique peuvent **réduire le flux sanguin utéro-placentaire** de telle sorte que l'ischémie ou l'hypoxie non seulement dans l'utérus lui-même, mais encore dans le liquide amniotique, le placenta et le foetus, peut induire une **acidose foetale combinée (respiratoire et métabolique)** en association à des aspects tout à fait typiques et marqués de **décélérations tardives** qui sont franchement **réversibles** de même qu'accompagnés par des **rebonds marqués de tachycardie entre les contractions, particulièrement après administration d'oxygène.** L'auteur l'a appelée: **«détresse foetale subaiguë»** parce qu'elle demande environ une demie à quelques heures pour que la vie du foetus soit menacée ou soulagée de ce type de détresse foetale.

En outre, en cas de complications maternelles sévères comme une toxémie et des désordres cardiopulmonaires avec ou sans modifications organiques du placenta, **«la détresse foetale chronique»** est fréquemment développée avec des signes de décélérations tardives du rythme cardiaque foetal légères ou modérées, sur une ligne de base légèrement élevée, d'irrégularité diminuée, qui apparaît insidieusement mais de façon **conséquente à chaque contraction** depuis le tout début du travail accompagnée par une **acidose métabolique foetale primaire,** qu'est médiocrement réversibe.

Par ailleurs, indépendamment de la présence de l'acidose intra-utérine ou de la bradycardie pendant le travail, certains nouveau-nés ne crient pas aussi vigoureusement que prévu, révélant qu'une **malformation sévère** ou une **lésion de naissance grave** comme une anencéphalie, une hernie diaphragmatique, une fistule oesophagotrachéale, une anomalie cardiaque ou une hémorragie cérébrale pouvait être la cause de la **«dépression organique»** à leur période périnatale. D'autres, qui développent une apnée et une dépression périnatale, sans qu'on puisse suspecter de telles étiologies, peuvent être considéré comme une **«dépression essentiell»** des centres respiratoires du foetus, le plus souvent due à des **dépresseurs narcotiques** ou un choc vagal survenu immédiatement dans les quelques secondes de vie post-natale.

Autrement dit, en fonction des caractéristiques dynamiques, selon la rapidité avec laquelle un foetus peut être menacé, nous avons maintenant, une **classification des dépressions périnatales en 5 catégories** qui sont: **essentielle, aiguë, subaiguë, chronique** et **organique,** lesquelles sont respectivement induites ou traitées en **secondes, minutes, heures, ans et mois.**

Bien plus, chacune d'entre elle correspond au dysfonctionnement de chacun des multiples circuits des systèmes de contrôle feedback de la vie, tels que les **sous-systèmes nerveux, circulatoire, respiratoire, métabolique** et **organique** qui doivent être explorés par leurs propres signaux spécifiques respectifs: électrique, mécanique, physicochimique, biochimique et biologique.

Par conséquent, il peut être facile de comprendre que ni le pH sanguin capillaire foetal, ni le rythme cardiaque foetal, ne peuvent isolément récouvrir tout l'évential du spectre diagnostic des dépressions périnatales de la vie, parce que chaque circuit de feedback a une dimension dynamique différente dans sa détérioration et son appartenance respective à l'un des cinq sous-systèmes de vie.

Mots-clés: Modèle de circulation foetale, pathogénie de la détresse foetale, acidose foetale, décélérations du rythme cardiaque foetal, classification des dépressions périnatales, cybernétique.

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